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## CHARACTERISTICS OF THE PROGRESS OF MALARIA IN WAR CONDITIONS

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Malaria is usually very prevalent during wars, and a number of peculiarities appear in its course. Because of this some authors (Heitmann) refer to it as war malaria. The morbidity rate of malaria was also noticeably increased during World War II, especially in the spring and summer months, when it was one of the most common of all diseases afflicting troops in the armed forces (Badyl'kes, 1943).

In connection with this it will be useful to analyze the cases of malaria among military personnel of N garrison in the first two and a half years of the war.

During this time, 1,281 persons stricken with malaria were treated at the garrison hospital, of which 473 (37 percent) were initial cases and 808 (63 percent) were recurrent cases.

Pl. vivax was discovered in the blood of 1,010 cases, Pl falciparum in 119 cases, and a mixed invasion (Pl.vivax plus Pl. falciparum) in 18 cases. Malarial parasites were not discovered in the blood of 134 (11.2 percent) of the cases in spite of positive clinical symptoms. All of the latter cases suffered recurrent malaria and had antimalarial treatment before they were admitted into the hospital.

It must be mentioned that many of the patients lived in various oblasts and republics of our immense country before their military service in N garrison. Inhabitants of the bleak North were, also present along with those of Siberia, the Baltic region, the Ukraine, the Caucasus, Central Asia, etc. There was an occasion to bring in various types of malarial parasites with different characteristics and virulence, which could not fail to develop specific clinical symptoms of the diseases.



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Malaria patients were admitted into the hospital all year long. However, the malarial curve began its ascent in April and reached its maximum in various summer months, differing from year to year.

In winter months the malaria cases were almost exclusively found in the surgical sections of the hospital; usually it appeared in patients after an operation or blood transfusion which often played a role in provoking malarial infection.

In the last years of the war the military malaria patients, as a rule, were treated in hospitals, as the military doctors tried to evacuate fever patients out of military sectors. Consequently, in distinction from peacetime, almost all malaria cases were concentrated in hospitals during these years, and there was no special choice of patients with serious visceral malarial affection.

In spite of this, 154 (12 percent) of our patients had serious forms of malarial affection of the internal organs and the central nervous system. Twenty-nine of these cases showed affection of the nervous system; 12 cases, renal affection; 24 cases, nonalbuminous edema; 48 cases, gastrointestinal affection; 12 cases, parenchymatous hepatitis; seven cases, avitaminosis; and 24 cases, cardiac affection.

The discovery of malarial parasites in the blood, unusual clinical progress of the disease, and also -- in the great majority of cases -- the effectiveness of specific antimalarial treatment were the bases for the diagnosis and etiology of malaria.

Affection of the nervous system was a comparatively frequent complication of malaria. Laveran had commented upon the high susceptibility of the nervous system to the malaria virus. The great psychiatrist, Krepelin, called malaria, "poison" (nerve poison). Other authors (Vyyasnovskiy, 1936; Myasnikov, 1936) indicated that malaria plays no smaller a role than syphilis in the etiology of neurosis and dementia. Kushev (1927) considered an affection of the nervous system as the most common malarial complication.

The malarial coma is the most dangerous of the nervous complications. We recorded the neuroparalysis in 16 patients: in 1941, one case (September); in 1942, three cases (also September); in 1943, 11 cases (four in August and seven in September).

In all cases, this coma condition developed in patients during the primary stage of tropical malaria, while 2-4 days to 2-3 weeks passed from the onset of the disease until the appearance of the coma condition.

The patients were admitted into the hospital in serious coma condition (usually on the first day of the onset of this condition); involuntary urination and defecation were observed in some cases. As a result of energetic antimalarial and symptomatic treatments, seven of 16 patients with malarial coma were saved and cured.

In studying the fatal cases of malarial coma, the presence of other serious complications which alone could result in death was discovered, in addition to the characteristic clinical and patho-anatomical degenerations for a given type of malaria. In four cases, the malarial coma developed as a complication of pneumonia, one case complicated with adenocarcinoma of the stomach, two cases were complicated with severe ulcerative tuberculosis of the lungs, and one patient had an acute hemorrhagic nephritis with manifestations of severe affection of the myocardium. In only one patient was death due directly to malaria, as no other disease was determined. This patient had been hospitalized after the third day of this coma condition.

Four of the malaria cases were complicated with psychosis: a depressed condition accompanied by hallucinations, sensations of fear, and delirious ravings. Three of these cases recovered while one of the patients in whom the psychosis persistently remained had to be discharged from the service.

Meningeal syndrome was a serious nervous complication in four patients. These patients were admitted into the hospital with symptoms of meningitis and were treated from the first day in the hospital with sulphide, but without success. Later, malarial parasites (in three cases Pl.vivax and in one case Pl. falciparum) were discovered in the blood of these patients. Atebrin treatment promptly relieved the acute symptoms of malaria and meningeal syndrome.

Other forms of neuromalaria which we observed were three cases of serious nephritis of the trifacial nerve and one case with epileptiform fits during malarial attacks. Atebrin treatment here also produced good results.

In comparison to a number of authors who recorded comparatively large incidences of acute nephritis in malaria patients (13.4 percent according to Sublin; 1.7-8.8 percent according to Klykov; 14.5 percent according to Shakhmatov), we discovered this disease in only 12 cases (0.9 percent), which can be explained by the early hospitalization of the malaria patients. In comparison with diffuse nephritis of a nonmalarial etiology, malarial nephritis was distinguished by a milder progress and much more favorable prognosis. In the time that a majority of cases of nonmalarial nephritis had passed through the edema type with an expressed tendency toward insufficient circulation of blood (Asabel', 1943), an edema syndrome sharply predominated in the clinical treatment of malarial nephritis, hypertonia was considerably less indicated and stable, and the symptoms of cardiovascular obstruction comparatively mild. In 13 percent of the cases of nonmalarial nephritis there was severe renal eclampsia, while in malarial nephritis this complication did not occur a single time. In the treatment of patients with nonmalarial nephritis, 45 percent of the cases were cured, but in the group of patients with malarial nephritis the disease took a persistent subacute progress in only one person.

We observed nonalbuminous edema, which was characterized by the development of edemas throughout the entire body without kidney affection (hypertonia, albuminuria, hematuria, etc.), more frequently than malarial nephropathy (in 24 cases). All the patients with nonalbuminous edemas recovered and were cured after a comparatively prolonged treatment (on the average, 26 days.)

Affection of the digestive organs was the most common complication of malaria in our patients. There were 48 cases with gastrointestinal complication and 12 cases with parenchymatous hepatitis.

The affections of the gastrointestinal tract were divided into the following clinical classifications: gastritis (of which 11 cases showed anacidity, 6 hyperacidity and two normal acidity), ulcerous stomach (four cases), acute enterocolitis (19 cases), and appendicitic syndrome (six cases).

Under the influence of hospital treatment all the patients with malarial gastritis, enterocolitis, and parenchymatous hepatitis recovered and were ordered back to their unit. Four malaria patients were admitted with aggravated ulcers which, apparently, were complicated by malaria. These patients had had ulcers for a number of years; then prior to the war the ulcers had stopped bothering them and they considered themselves cured. In the beginning of the war they were mobilized and served for one and a half years in the Soviet Army. Several weeks after the appearance of malarial paroxysms, strong pains occurred in the epigastric region. Laboratory examination closed Pl. vivax in their blood and Gaudek's recess (Nishe) in the stomach of in the duodenum.

We consider the pathogenic role of malaria in causing aggravated ulcers entirely possible, as pathological degeneration in all parts of the vegetative nervous system (Mogil'nitskiy, 1936) are observed in malaria, and it is known that affection of the system has substantial significance in the pathogenesis of ulcers.

Professor Gefter, in his report on the Interoblast Conference of Therapeutists (Gor'kiy, 1946) also noted the capacity of malaria to aggravate ulcers and, in particular, to provoke a hemorrhage from the ulcerated mucous membrane of the stomach.

Laveran (1901) also remarked that vomiting, accompanied by severe gastric pains and hemorrhage, and dyspepsia can be complications of malaria.

As a result of a combination of antimalarial and antiulcer treatment, the sharp manifestations of malaria and symptoms of aggravated ulcers (including the Gaudek recess) disappeared in all patients after 3-5 weeks.

There were positive symptoms of acute appendicitis accompanied with high temperature and the presence of Pl.vivax in the blood in six of the patients admitted into the hospital. These symptoms quickly disappeared in four of the patients through active antimalarial treatment. From this it may be assumed that the so-called malarial appendicular colic appeared in these cases. In the remaining two cases, symptoms of acute appendicitis complicating active malaria were sharply indicated and leukocytosis was observed in the blood, with a shift of the leukocyte count to the left and toxigenous granularity of neutrophils. It was necessary to operate on the patients immediately. Suppurative appendicitis was discovered in the operation. The latter cases make it necessary to be on the alert in the presence of appendicular syndromes in malaria patients; careful simultaneous supervision of therapeutics and surgery is necessary here.

Seven of the malaria cases were combined with strongly expressed endogenous avitaminosis (two cases with scurvy, three with pellagra and two with polyavitaminosis -- scurvy, pellagra, and hemeralopia). Avitaminosis developed in malaria case which were poorly treated (1-3 months after the onset). The increased demand for vitamins in malaria, as well as the affection of the gastrointestinal tract and the liver caused by this disease which makes the absorption and assimilation of vitamins difficult, aided the development of vitamin deficiency. The research of Iareyev (1943) and Kuperman (1945) which established a considerable deficiency of ascorbic acid in the organisms of malaria patients confirms this.

Vitamin therapy of malaria patients suffering with avitaminosis was ineffective before the curbing of malaria with specific treatment. Only after these treatments were we able to bring about a relatively quick recovery in the patients (10-34 days) by prescription of the appropriate vitamins and hence return them to service. Our material has shown that a comparatively frequent affection of the myocardium was one of the various peculiarities observed in the progress of malaria in wartime. Out of all the malaria patients who were treated at the hospital 24 (approximately 2 percent) were admitted with an active symptom of poor blood circulation. Seventeen of this group had not had any symptom of the cardiovascular system prior to falling ill with malaria, and there were only five cardiac patients and myocardiosclerosis in two patients; but these afflictions prior to the development of malaria were in an arrested stage and the patients considered themselves entirely fit for service.

Clinical symptoms of cardiovascular obstruction usually developed several weeks or months after the appearance of the first malarial paroxysms. The development of cardiovascular obstruction in malaria is due to dystrophia myotonica of the myocardium. Heavy physical strain, to which the soldiers were subjected in the severe years of the war, and anemia caused by malaria (the volume of hemoglobin in our cardiopaths was 29-40 percent) contributed to the development of dystrophia myotonica of the myocardium. As a result, the myocardium -- affected by the malarial process (according to Bin'yami, parasitic thrombus was discovered in the capillaries of the heart in serious cases) and insufficient amount of hemoglobin because of anemia -- became weak, and the patients were admitted into the hospital with more or less serious symptoms of poor blood

circulation. The treatment of these patients with cardiotonics without simultaneous use of antimalarial medicine was ineffective. Only active antimalarial treatment (in conjunction with cardiotonics, and sometimes without them) normalized the blood formation in a comparatively short time (2-6 weeks), and restored them to service. As a result of the treatment, we succeeded in returning 20 out of 24 cardiopaths to the army.

Combinational forms of malaria were observed only in connection with various infectious diseases of the respiratory organs: 12 cases of pneumonia, one case with an abcessed lung, and four cases of tuberculosis of the lungs.

The pathologist-anatomist Shirokogorov (1939) confirmed the fact that malaria patients are especially predisposed to croupous pneumonia. We observed malaria complicated with this disease in only three cases. Malaria was complicated with bronchial pneumonia more frequently (nine cases).

The progress of malaria complicated with pneumonia was almost always very severe. In spite of active treatment with antimalarial agents and sulfidine, 8 out of 12 pneumonia patients died, and four of these developed a coma before they died. Only four malaria patients recovered after a month and a half of treatment.

Similar to Shirokogorov, we discovered leukopenia (the leukocyte count was 3400-5500) in malaria complicated with pneumonia, which the author considers one of the chief causes of death in the majority.

We are adding to malariology the unfavorable progress of mixed infection of malaria and tuberculosis of the lungs. We observed such a mixed infection in four patients. All of them suffered 3-day malaria. In only one of the patients was tuberculosis in an arrested state, despite a considerable duration. In the remaining patients, tuberculosis of the lungs progressed with great destruction. In two of the latter group, tuberculosis apparently was responsible for the development of comatose condition and caused death.

Thus, we cannot agree with the opinion of Shirokogorov, Boudin, Veselko, and Bin'yami on the immunizing influence of malaria against tuberculosis.

The basic antimalarial preparation during World War II was atebrin. 1,167 persons (91 percent) were treated with this preparation, while only 114 (nine percent) were treated with quinine.

Our experience with the treatment of malaria patients with atebrin once again confirmed its high effectiveness. With atebrin treatment of the noncomplicated and noncombinational forms of malaria, the period spent in the hospital was on the average 7 - 12 days, and the patients were ordered back to their units. Forms of malaria which did not respond to atebrin treatment were observed in only four cases, who were successfully cured with quinine. Of 1,281 malaria patients, 1,256 (98.1 percent) were cured; 11 (0.9 percent) were declared unfit for military service. Various accompanying diseases (cardiopathy, myocardiosclerosis, tuberculosis of the lungs, results of a war wound, etc.) were causes of disability. These diseases had already made them unfit for service, and malaria only hastened the progress of these diseases. Only in singular cases (malarial psychosis, malarial subacute nephritis) was malaria the cause of disabling patients for a long period.

Thirteen patients (1 percent) died. Two persons, one of them suffering from hemorrhagic nephritis, died in a comatose condition directly from malaria. In the other cases serious diseases with which malaria was complicated contributed

to death: eight cases of pneumonia, two cases of tuberculosis of the lungs, and one case of cancer of the stomach.

## CONCLUSIONS

1. During World War II malaria was distinguished by the variety and seriousness of types affecting the internal organs.

2. The most frequent visceral affections in wartime malaria were: malarial infection of the nervous system, myocardium, digestive organs, kidneys, and nonalbuminous edema.

3. With timely antimalarial treatment, all types of visceral malarial affections, including malarial coma, recovered comparatively rapidly if the malaria was not complicated with other diseas s which could cause death (tuberculosis, cancer, pneumonia, etc.).

4. The experience in the use of atebrin for treatment of several hundred malaria patients, a considerable number of whom suffered serious visceral affections, showed that this preparation is very effective.

With the correct introduction of atebrin treatment, all types of malaria were cured and the patients recovered.

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